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EDITORIAL

THE NEW YEAR

WITH the issue of this number of THE BRITISH VETERINARY JOURNAL a new year opens. We take this opportunity of wishing our readers the familiar wish—a happy New Year. Before another year is ended let us hope that we will have reached a period when we can look back and say that much has been accomplished for the well-being of our country, our Empire and the whole world. Strenuous times still lie ahead, but with goodwill and mutual understanding we are bound to accomplish much throughout the world.

NEW TITLE—THE BRITISH VETERINARY JOURNAL

OUR readers will note that with the start of the 105th volume, the title of *The Veterinary Journal* has been changed to THE BRITISH VETERINARY JOURNAL. It has been felt for some considerable time that the word "British" should be included in the title. The "Journal" has a wide circulation throughout the world and is read by veterinarians and others in many countries. As the "Journal" is produced in Great Britain and contains contributions very largely by veterinary surgeons and others within the Empire, the inclusion of the word "British" in the title will mark the origin of the publication. In adopting this new title, the *Veterinary Journal* is falling into line with a number of scientific publications dealing with allied subjects. It has always been the aim of the editors and publishers to ensure that veterinary interests in the Empire have been maintained and that the *Veterinary Journal,* published in London, the heart of the Empire, compared favourably with journals of a similar type from other countries. THE BRITISH VETERINARY JOURNAL will, we hope, continue to fulfil this purpose.

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UNIVERSAL COLLABORATION

SCIENCE knows no territorial boundaries: discoveries by workers and observers in any country are free to the whole world, and the results, with the necessary modifications, are open to all for application. This is certainly true of veterinary science, examples of which are too well known to need quoting. It may interest our readers to know that recently a meeting of veterinary workers from a number of countries in Europe took place in Warsaw, where exchanges of ideas were made and the results of investigations on some of the important diseases of livestock were freely discussed. The meeting was arranged by the Food and Agricultural Organisation of the United Nations, which was represented by veterinary surgeons from America. Much credit is due to the organisers for the able way in which all the arrangements were carried out and for the success of the meeting. Great Britain was among the countries represented and we took our share in the talks, demonstrations and discussions. A meeting of a somewhat similar type was held in this country last year, when representatives of many European countries met and heard of the progress in disease control methods which had been studied and devised during the past ten years or so.

Because of war conditions it was not possible for all countries to continue with research activities: it is therefore the duty of those, so fortunately placed that their work has continued during the war years or has even been extended, to acquaint their less fortunate colleagues with the advances that have been made. The meetings now referred to were convened largely with that object in view. It is true, of course, that many results have been published in our journals and that the circulation of scientific literature is now beginning again. Those unable to obtain the journals during the war have missed much. Again, while we all take pride in the manner in which we try to present our results, personal explanations and demonstrations are the surest methods of "getting over" the results and discoveries. In a short space of time the different points are made clear and the difficulties of technique are overcome. It was quite obvious that these meetings fulfilled the purpose for which they were arranged.

Following a war in countries engaged in active military and other activities, the health of livestock must always suffer. Disease cannot be controlled in the successful way carried out before the war took place and new diseases are almost certain to be introduced. In such countries the veterinary personnel have an immense task to carry out: they have to devise the best measures to cope with the problems which now face them. Those of us who have the knowledge and experience of new methods of diagnosis and control have our part to play in imparting to our colleagues in these countries all the information we have accumulated on subjects which might be of value to them. At these meetings all the available knowledge was freely given and freely discussed.

The Food and Agricultural Organisation of the United Nations has done a noble bit of work in different countries throughout the world in so assisting with veterinary problems. Such assistance is to be encouraged to the fullest extent. It is well recognised that the health of livestock is one of the essential

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requirements for increasing our food production: this is seen in the individual country—it is of even greater importance throughout the world as a whole. By this form of collaboration, universal in character, we can play our part in the drive for food: by the efforts of an organisation like F.A.O. much can be accomplished. Let us, who can give, continue to disseminate the knowledge we have accrued for the benefit of those who have not had the opportunities for work accorded to us.

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GENERAL ARTICLES

OBSERVATIONS ON SPASMOPHILIA IN DOGS, INCLUDING CANINE HYSTERIA*

By TOM HARE, M.D., B.V.Sc., M.R.C.V.S.

THE mistaken notion is still current that the American authors of 30 years ago, who invented the names *canine hysteria* or *frights' disease,* had discovered some unrecognised disease or syndrome. During the 19th century, French and British authors described fits objectively and separately, according to the pattern of behaviour, as epilepsy, eclampsia (epilepsy during pregnancy), megrims, vertigo, staggers, tetany, etc. They attributed the fits to (1) functional (i.e., unrecognised) causes and (2) irritation, by parasites or chemicals, either of the alimentary tract or of the exterior of the body. They regarded the fits as disease of the central nervous system, but they left its pathology in obscurity. The American authors, as a result of their chemical studies of the patient's diet and body tissues, recognised fits as the expression of disease of the whole animal and not of the nervous system alone.

This idea of a morbid process, in the course of which fits are merely spectacular events, is my theme to-day. I am presenting a summary of my records of 58 cases in dogs of conjoint infection by the *beta hæmolytic streptococcus* (B.H.S.) and a bowel parasite, in the course of which one or more fits occurred. The fits, though differing in the pattern of their expression, had one characteristic in common, viz., involuntary contractions of muscles. Thus, under the old term *spasmophilia,* which is defined as a tendency to attacks of involuntary contractions of muscles, both the illness and the fits can be integrated. I have not arrived at my present views on spasmophilia without mistakes and hesitations. When I wrote a letter to the *Veterinary Record* (1938, *50,* 348), I was approaching the symptomatology of canine hysteria subjectively and trying to correlate it with my pathological findings; I failed. When writing a recent paper on the B.H.S. infection (1946), I was reluctant to abandon the generally accepted view that canine hysteria is a specific disease.

My clinical study of the patient included a bacteriological examination of tonsil, vagina or prepuce swabs, and urine, a chemical and microscopical examination of urine and fæces, and, in some cases, a microscopical examination of the blood. Approximately half the 36 clinical cases have been followed up over periods varying from three to seven years. Twenty-two cases came to me for autopsy.

Some facts of the earlier history of 14 cases, which are not commonly obtained in consultant practice, have been tabulated below. They throw light on the pathogenesis of spasmophilia. The B.H.S. had been diagnosed by the veterinary surgeon in attendance and confirmed bacteriologically at the clinical onset in four cases, or at the dog's purchase in ten cases (the age of those in the latter category is preceded by a minus sign). In cases 2 and 12, the first fit occurred after the clinical diagnosis but before the results of the bacteriological examination of swabs was known. The table also shows the ages at the diagnosis

* Presented to the meeting of the Central Veterinary Society, May 6, 1948.

Ibid.: *Brit. Jour. Dermat. and Syph.,* 1946, *58,* 286.

of the bowel parasite, the occurrence of the first fit and at my first clinical investigation or autopsy (cases 6, 8 and 13). It will be seen that *at or before* the occurrence of the first fit, the diagnosis had been made of the B.H.S. infection in all 14 cases and of the bowel parasite in eight cases. Thus it would appear that the conjoint infection by the B.H.S. and bowel parasite had been established before the occurrence of the first fit.

Table showing the ages of fourteen spasmophilic dogs at: (c) diagnosis of the B.H.S. infection, (d) diagnosis of the bowel parasite, (e) occurrence of the first fit, and (f) consultation (clinical or autopsy).

a	b	c	d	e	f	g	h	i
		Age in months at						
Case number	Sex	Diagnosis of B.H.S.	Diagnosis of parasite	First fit	Consultation	Number of fits	between c. and f. Bowel parasite	Anti-distemper vaccine
1	♀	— 1½	1½	2¾	4½	Many	C.T.A.	Nil
2	♂	— 3	3	3	60	Many	C	Nil
3	♀	1½	4½	4½	5	1	C	+
4	♀	— 3	24	4½	24	1	C	+
5	♀	— 4¾	6	5½	6	8	C	+
6	♀	— 4	5	6	6	1	C	Nil
7	♂	6	6	6½	7	3	C	Nil
8	♂	Whelp	7	7	7	3	T	Nil
9	♂	— 6	9	8	9	23	C	Nil
10	♀	— 3½	3½	9½	12½	Many	C	+
11	♂	— 6	10	13	18	Few	C	+
12	♂	18	30	24	30	Few	C	+
13	♂	— 6	30	28½	30	4	C	Nil
14	♂	— 4	36	32	36	1	C	+

NOTES.—Column c: Date preceded by minus sign is also date of purchase.
„ f: Dates at which 6, 8 and 13 came to autopsy.
„ h: C = coccidia; T = roundworms; A = hookworms.
„ i: + = Burroughs Wellcome method; Nil = Either no vaccination or method not recorded.

The 14 tabulated cases were a representative sample of the age at onset of the first fit, which in 57 cases ranged from 11 weeks to three years; in one case the first fit was alleged to have occurred at 11 years of age. No significance attached to breed, sex and protection against the virus of canine distemper.

Pathology of Spasmophilia

The bowel parasites comprised coccidia, roundworms (*Toxascaris* or *Toxocara* sp.) or hookworms (*Ancylostomum caninum*). Occasionally one species of worm was found, with coccidia. From three cases, all three parasites were recovered.

In the earlier stages of spasmophilia the only abnormality found in the urine (excluding the cases with B.H.S. pyæmic nephritis) was a trace of albumen. Later, the urine showed evidence of degeneration of the liver (hepatosis) and kidneys (nephrosis), viz., a larger quantity of albumen, bile salts, granular epithelial cells, rarely tubular casts and no inflammatory cells. In the more advanced stages the urine also showed bile pigment, and blood in coccidiosis cases.

In the 22 fatal cases, autopsy revealed a combination of the pathology of the two causal infections. In the time available, I can mention only a few main features of the pathology of the B.H.S. infection, which has been described by Dr. R. M. Fry and myself (1938 to 1946), and that of coccidiosis and bowel-worm infections, which has been described by Joest (1926). The essential injury produced by the B.H.S. toxin is of the reticulo-endothelial system in the form of a degeneration of endothelium and hæmolysis; that produced by a bowel parasite is a degeneration of epithelium of the small intestine and liver. In brief, autopsy revealed anæmia, œdema of the lungs, emaciation (usually), chronic tonsillitis, catarrh, hyperplasia of the cervical popliteal and mesenteric lymphatic glands, slight hyperplasia of the spleen, chronic catarrh of the pylorus of the stomach and proximal third of the small intestine, some terminal capillary hæmorrhages in the pylorus of the stomach, nephrosis, albuminous degeneration of the liver and the toxic encephalopathy of the brain, which was described by the late Professor John Eyre (1928). Protracted cases also revealed varying degrees of repair, e.g., fibrous deformity of the tonsils, fibrosis of the pylorus and duodenum, cirrhosis of the liver, fibrosis of the kidneys, sclerosis of the brain (to which I shall return later in the paper). I have interpreted these degenerative changes as those of an intoxication or poisoning of the whole body.

Symptomatology Before and After a Fit

I shall describe the symptomatology before and after a fit, leaving to a separate section the patient's behaviour during a fit. The various phases of the symptomatology of the B.H.S. infection have been described in previous papers. To these are added the signs of the particular bowel infection. The usual signs of the B.H.S. infection in the spasmophilic patient are those of tonsillitis with catarrh and enlargement of the cervical, popliteal and possibly other lymphatic glands, plus the parasitic infection of the bowels. Invariably the patient is anæmic. Not infrequently capillary bleeding may be observed in the sclera, gums, vagina and fæces. In a majority of patients some degree of emaciation is seen by the onset of the first fit. There is little variation of the temperature on either side of normal other than the usual rise to about 103 deg. at the intermittent recurrences of the B.H.S. tonsillitis. Temporarily the temperature may fall to 99 or 98 deg. during vomition and mucoid diarrhœa. Mucoid stools, intermittent or constant, may be associated with the expression of pain on deep palpation of the epigastrium, particularly over the duodenum. Diarrhœic stools may be melænic or tinged red with blood. Tympany commonly ensues, though it is of variable quality. The patient may secure temporary relief by erructation or flatus; the odour of the breath and flatus resembles that of a mixture of decomposing protein and butyric acid. In neglected cases, the stomach and whole small intestine may be tympanitic and the patient appears to be unable

to obtain durable relief by erructation or flatus. The distension of his abdomen is associated with restlessness and intermittent whimpering and yawning. In cases of coccidiosis the urine may be intermittently blood-tinged, or the last few drops may be blood red. Some form of B.H.S. dermatitis with pruritus is common.

The patient's expression, bearing and behaviour change. He may become quiet, inert, lethargic, somnolent, subdued, passive and unresponsive to his owner's offer of play. He may show no sexual response to bitches in œstrus; or she, when in œstrus, ignores and even resists the male. Here I would strongly deprecate the cruel practice of forcibly restraining the resisting spasmophilic bitch in order that coitus be secured. The spasmophilic patient may be careless of his training; he may urinate or defæcate against furniture without making any attempt to leave the room. He shuns or slinks away from strangers, probably snarling and snapping. At walk in the street, he avoids and may yap at passers-by. He may fly at and snap at his owner. His appetite may increase to voracity; usually it is depraved (pica) and he will eat grass, cinders, builders' refuse, fæces and carion, or he will lick the base of lamp-posts and pavements. The pruritus which characterises the chronic B.H.S. infection (rubbing of the back, loin and tail against furniture and the licking or nibbling of the feet, metacarpals and metatarsals or prepuce) may persist and complicate the picture. The patient may masturbate.

The Spasmophilic Attacks or Fits

Before describing some examples of particular patterns of fit, I would offer some general observations on the spasmophilic attacks. Their onset is sudden and unpredictable, but there is considerable variation in their duration, quality, pattern and number, both in the one patient and as between different patients.

The sudden *onset* is associated, not infrequently, with some happening, which may be either (a) external to the patient, e.g. the backfire of a motor, the rattle of the patient's food plate, the approach of a stranger, the appearance of a light during darkness, or (b) of the patient's own making or inner environment, e.g. "cocking his leg" to urinate, defæcating or passing flatus, during rest in the home, sleep, play, racing on the track, transport, before or after a feed, after eating garbage, etc. But the onset of every attack in the same patient is not associated with the same, or even with a comparable, happening. Moreover, an attack cannot be induced by deliberately reproducing the happening. It would seem that these happenings, which loom large in the owner's mind, are coincidence and of no etiological significance.

The *duration* of the fits varies from about 5 to 10 seconds in the mild to about 2 to 3 minutes in the violent or convulsive fits. The timing of the fit in a patient which has galloped out of the observer's sight is very difficult, if not impossible; also, the estimate of an owner who has been distressed and preoccupied by attempts to catch the patient can be accepted with sympathetic reserve. The timings accepted for this paper are my own or those of observers in whom I have confidence. I would point out that the duration of the violent fits is some 30 or more times that of the mild fits.

From studying the patient and his history, I have been unable to predict

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the recurrence or number of fits, which may occur before treatment becomes effective or death ensues. In this matter the biochemist may eventually establish criteria for prognosis. Only one fit was observed in case 14 (see table), a rather severe epileptiform fit, although specific treatment did not begin until four months later. Twelve months after treatment began, he was the best gun-dog in his county and had had no recurrence. Case 6 had B.H.S. tonsillitis when purchased at 4 months; at $5\frac{1}{4}$ months coccidiosis was diagnosed; specific treatment began at $5\frac{1}{2}$ months; at 6 months he had an epileptiform fit and about 48 hours later he passed into "acute hæmorrhagic gastro-enteritis," uræmic convulsions and death; autopsy revealed B.H.S. pyæmic nephritis and coccidiosis. Case 3 had an ambulatory fit at 3 months; his second, a violent ambulatory fit, occurred at 3 years. Subsequent fits were epileptiform, the third at $3\frac{1}{2}$ years, the fourth occurred three weeks later, the fifth at 3 years 10 months, after which he had numerous fits until specific treatment began at 5 years.

The *mild* spasmophilic fits have slight, if any, influence in deflecting the patient from his occupation. Their after-effects, if any, are fleeting. There is no noticeable initial tonic spasm. Sweating, salivation and involuntary micturition or defæcation are rarely observed and then are but slight. One of the mildest patterns of spasmophilic fit is the *vaginismus,* which may occur on passing a swab. The fine clonic spasms of the vaginal wall arrest the swab at the meatus urinarius for some 5 to 10 seconds; at their ceasing, the swab can be passed up to the cervix without further obstruction. Similarly, involuntary clonic spasms of the anal and rectal muscles may obstruct the passage of the thermometer or finger into the rectum. These brief attacks of involuntary spasms of vagina and rectum differ from the voluntary contractions and relaxations of the resisting patient. The latter are coarse, expulsive and involve the pelvic, and even the back, muscles, and the hind legs are partly flexed.

Tremors (trembles, shivering fits) are characterised by small and rapid involuntary spasms which are rhythmical in time and force. To the observer's sight commonly they appear to be unilateral and to involve one muscle or a group, e.g., the upper lip (lip twitch), neck, back (resulting in lateral curvature) or a limb. Examples of bilateral tremors are "head nodding" due to spasms of the flexors of the neck, "teeth-clacking" due to spasms of the masticatory muscles, "flopping on to the tail" due to spasms of the flexors of the thighs. Tremors of the adductors of the thighs result in the hind legs being brought together or crossed. When examining patients during tremors, I have formed the impression that the spasms involve more muscles than is apparent to the eye. For instance, during apparent unilateral tremors of the upper lip of a dog my hand, which was lightly compressing his tracheal region, distinguished clonic spasms of the peripharyngeal muscles. In another case, while palpating her perineum, a bitch had an attack of tremors of one flank and I could feel the clonic spasms of the muscles beneath pubis and ischium bilaterally. I had my finger in the rectum of another patient when he had tremors of one shoulder and I could feel the contractions of the rectum and the anus. The more forceful types of tremors, though beginning in one muscle, spread to many muscles, unilaterally or bilaterally. For example, the "lip-twitch" may rapidly spread

to the muscles of mouth, face and neck, like an incoming wave. The same muscles or group of muscles is not necessarily affected by each attack of tremors. If at the onset one of my patients was standing erect, the contractions occurred in the flexors of the hind limbs and she would sink onto her rump; whereas if she was resting on her sternum, the contractions occurred in the muscles of one shoulder and side of thorax and she would sink into the prone posture. During tremors the respiraton rate may be increased, and in the more forceful attacks the patient may pant.

In another group of milder fits, the involuntary spasms are masked by the patient's gross movements. If at the onset of a unilateral spasm of the neck the patient is standing erect, he may circle, " chasing his tail "; usually he falls onto one quarter with the opposite hind leg fully flexed at the femoro-tibial joint, abducted at the hip and in tremor. During the fit he may be silent or yapping. A variant of this type of attack is the " sledging fit," in which the patient suddenly circles, biting at his flank, squats on his rump, extends his head and neck and, by a series of rapid jerks of his back and abdominal muscles, sledges forwards on his rump.

In *tetany,* either the total effect of the contractions appears to be greater than that of the relaxations or the action of extensors is greater than that of flexors, or vice-versa. For insance, one patient at the onset of tetany was running; suddenly his two forelegs were flexed at the carpus, he knuckled over and fell; the flexion at the carpus ceased after about half a minute and he rose to his feet. Another patient, when walking in my laboratory, had an attack of tetany involving the adductors of the thighs; he rocked and fell. On lifting him up, I noticed that both forelegs were affected by clonic spasms, though those of the extensors were stronger than those of the flexors, resulting in the forelegs being raised to the level of, and parallel with, the neck. Incidentally, these spasmophilic attacks can be differentiated easily from the behaviour of the patient, which, owing to weakness, loses his balance and falls on being turned sharply through 360 deg.

The violent or convulsive types of fits put a stop to whatever the patient was doing at their onset. They are followed by a stage of recovery, the duration of which is proportionate to the quality of the convulsion. Violent fits can be sub-divided into *ambulatory* and *epileptiform,* according to whether the patient moves or retains his station during the fit.

Ambulatory fits (megrims, hysteria, frights disease, running fits). At the sudden onset of the fit, the posture is changed; if the patient was moving, he stops abruptly; if in the prone position, he rises to his feet. Thus the initial posture is the erect. The whole body is tautened in tonic spasm; head and neck are extended; ears, lips and eyelids are retracted; the mouth is shut; eyeballs are fixed in a forward stare; the hairs of the dorsum are erected; the tail is extended and the muscles of respiration are fixed. The tonic spasm is of fleeting duration and is followed by rapid contractions and relaxations, which appear to involve all the muscles of the body. According to the force of the clonic spasms, the patient progresses at a walk, run or gallop, emitting a succession of cries or howls. Occasionally forward progression is preceded by a few steps

backwards. There is considerable variation in the speed, the course taken and the distance covered by the patient. He may walk or run into a dark corner of the room or gallop on a circular, irregular or straight cross-country course, over, under or around obstacles. He may start on a circular course and suddenly dash off at a tangent, leaping the garden wall and galloping down the road; or he may start on a straight course and end on a circle. The onset of the first fit in case 13 occurred while he was playing with another dog in the garden; he bolted into the house yapping, up the stairs, leaped at a bedroom window, bounded off the glass pane onto the floor, crouched and sprang into the wash-stand basin, where he stood shaking all over, with nose up, howling. This case exemplifies highly elaborate movements performed with power, precision and efficiency; a remarkable performance for the untrained muscles of a dachshund. During an ambulatory fit the whole dog appears to be energised; at its cessation, the whole dog is depressed, and the degree of depression and fatigue appears to be proportional to the duration and quality of the attack.

During an *epileptiform fit* (epilepsy, eclampsia, convulsions) the patient remains in his station. At its sudden onset, he stiffens in a tonic spasm; his head is retracted; the jaws are clenched; the eyelids are retracted, exposing the sclera; the pupils are dilated; the limbs are extended; the back is over-extended; the muscles of respiration are fixed. If the patient is standing at the onset of the fit, he falls. The tonic spasm is replaced by a succession of clonic spasms, which vary in intensity. The unconscious patient is prone on his side; his limbs are "paddling" or kicking; the respiration rate is increased; he may be yapping, crying, yelling or uttering staccato yaps. The patient salivates, "foams at the mouth"; he becomes cyanosed, the "tongue turns blue"; usually sweating is noticeable; unless the bladder and rectum are empty, urine and fæces may be passed. With the passing of the convulsion the patient becomes flaccid, respirations are deep and either recovery sets in or he passes into semi-coma and death. I am using the term *recovery* from a fit as signifying resumption of the pre-fit behaviour.

Sequels To a Fit

(1) The resumption of the pre-fit behaviour is immediate on the cessation of the mild fit; it is delayed after a violent fit and the patient is fatigued or exhausted. In his fatigue, lasting a variable period of 10 to 60 minutes, he readily accepts his owner's caresses, comfort and warmth. He may sleep. For some hours after a violent fit the muscles, particularly of the back, appear to be tender on palpation.

(2) There is no resumption of the pre-fit behaviour. The patient may recover consciousness for a shorter or longer period, and may even accept some liquid nourishment, but further convulsions occur and he passes into coma and death.

(3) I have not witnessed the cutting-short of a violent fit. In consultation with Mr. Denys Danby, M.R.C.V.S., I examined a pointer, aged 2 years, which one month previously had a violent ambulatory fit in Regents Park. About one hour later, when walking beside the lake in the park, a second fit began. The patient leaped onto the thin ice, which broke. The dog was immersed, came

to the surface and, paddling to the edge, hauled himself out. From the owner's description and timing of the events, we inferred that the duration of the fit was shortened by the ducking, and that there was no period of recovery as after the first fit.

Treatment of Spasmophilia

Specific treatment of spasmophilia is directed against its conjoint causal agents. The treatment of the B.H.S. infection has been described in previous papers. Tetrachlorethylene (P.D. & Co.'s "Nema Capsule"), repeated on the tenth day, is advised for roundworms and hookworms. Preferably, a gastro-intestinal lavage with warm normal saline or glucose saline should be given 24 hours after each dose of the vermifuge. For coccidiosis, I advise "sulphamerazine" (P.D. & Co.), half a gramme at eight-hourly intervals for seven days, to be followed by three days without the drug; this ten-day cycle is repeated for 30 days, at the end of which the fæces should be examined. Sulpha drugs give better results in acute than in chronic coccidiosis, for which a course of "sulphamerazine" may be alternated with a course of pulv. ipecac. co. grs. X daily for 12 days, to be followed by seven days without the drug before repeating the 12-day course.

For *symptomatic treatment* of the intoxication, I advise gastro-intestinal lavage with sodii bicarb. (one teaspoonful to the pint); patients with tympany may require lavage on three successive days. Lavage is followed by mist. sodii citratis, bicarbonatis et salicylatis, grs. V, *aa.*, b.i.d., together with glucose and barley water *ad lib* as drink. On clearance of the bowel parasite and mucus, ferri et ammon. cit. is given. Vitamin therapy does not appear to confer any measurable benefit on patients which are receiving good food and nursing. Until the bowel parasite has been cleared out, the patient should be confined to kennels. In order to reduce the risk of reinfection by the bowel parasite, strict sanitary measures are essential; each day the fæces should be removed and burned and the kennel should be scrubbed thoroughly with hot water and disinfectant. Narcosis contributes nothing to the patient's benefit and is justifiable only as an emergency measure, e.g., when the patient's recurrent fits are distressing an owner already overstrained by anxiety and disturbed sleep.

Comments on treatment. A brief reference to one of my periods of perplexity may explain the importance I attach to the order in which the various treatments should be administered. In the earlier days of my study of spasmophilia I found that if the B.H.S. vaccine was given *before* the bowel treatment a significantly large minority of patients developed a spasmophilic fit, of varying quality, almost immediately upon the injection of the vaccine. I attributed the phenomenon to hypersensitivity to the B.H.S. antigen until I found that it could be induced by injecting 2 c.c. sterilised peptone broth or milk. Also it became apparent that a fit might be induced at the second, or even at the third, dose of vaccine only in patients whose bowel treatment had been delayed. By withholding the administration of vaccine pending treatment of the bowel a fit was not induced. Thus by a process of elimination I came to advise that we should first treat the bowel infection (with the specific anti-parasite drug, lavage,

mist. sodii, nursing) and then after an interval of four or more days inject the first dose of the B.H.S. vaccine.

The duration of treatment requires consideration. The removal of one of the two causal infections suffices for cure of the spasmophilia though *not* for cure of the patient. Provided adequate sanitary measures have been taken against reinfection the removal of bowel worms is usually effected within two weeks. Cure of an initial acute coccidiosis may be effected in about three to four weeks; the treatment of the chronic infection can be more protracted. We need a large-scale controlled evaluation of the available methods of treating coccidiosis; under clinical conditions it is not easy to assess how much of the delay in cure is due to defects in the drugs and how much to inadequate sanitary measures; I have the impression that reinfection is the major cause of delay in the cure of coccidiosis. Cure of the B.H.S. infection, by present methods, is inevitably protracted. Penicillin and/or the sulpha drugs are invaluable for treatment of the B.H.S. invasion of tissues, but they are mere alleviatives of the all too prevalent toxæmia which results from multiplication of the B.H.S. on mucous surfaces, e.g., tonsils. It seems to me that the best we have achieved so far is to neutralise the injurious effects of the B.H.S. infection by building up an immunity sufficient to prevent invasion of, and damage to, the internal organs. Usually this can be achieved by administering quarterly courses of the B.H.S. vaccine over a period of twelve months. But for the maintenance of that standard of immunity further courses of the vaccine at six-monthly intervals are necessary. The treatment of spasmophilia may be a matter of weeks, but the treatment of the patient is a long-term policy.

Prognosis

The basis of prognosis is a shrewd judgment of the quality of the poisoning. Upon the evidence of a fit neither the occurrence of further fits nor their quality can be predicted. Since much of prognosis is implied in what has been said already I will confine my remarks to one particular difficulty in assessing the evidence. Injury of the central nervous system is the result of (a) the spasmophilic poisoning, alone or combined with a meningo-encephalitis due to invasion by the B.H.S., or (b) repair, resulting in the over-production of scar-tissue (sclerosis). This method of classifying injury of the central nervous system is helpful at autopsy but beyond my clinical aptitude when examining the patient in life. Clinically I can recognise injury of the central nervous system which results in the durable loss of neurons as expressed by the neurological signs of localisation, altered reflexes, spasticity and paralysis. If I fail to observe such signs I conclude there has been no durable loss of neurons and I wait and see what happens. The signs of durable loss of neurons may appear (1) early in spasmophilia and before any fits, (2) after one or more fits, and (3) after the spasmophilia has been cured. I am directing special attention to this third type of occurrence because it would seem that the destruction of neurons is the result of exaggerated repair (sclerosis of the central nervous system). The possibility of its occurrence weeks or months after the patient has been discharged as cured of spasmophilia should not be overlooked; and the owner should be warned that some months must elapse before the possibility of

permanent defect of the brain can be excluded. Nevertheless, the appearance of neurological signs of durable loss of neurons, provided they do not interfere with the dog's functions and well-being, is not necessarily an indication for euthanasia. If they extend to more and more parts (progressive sclerosis) our opinion would be reviewed; if they remain fixed they can be regarded as the grave-stone of departed disease, a disability comparable to the shortening of a healed fracture. A familiar example of a localised durable loss of neurons is the *permanent* rhythmical spasms (choreiform jerks, chorea) of a muscle or group of muscles. By inducing the patient to fix his attention (e.g. on a cat), the spasms may be reduced in intensity for about a minute, but they never completely disappear. Their permanence distinguishes them from the involuntary spasms of the spasmophilic fit, before and after which the affected muscles function normally. Of my 58 cases of spasmophilia, on which this paper is based, I have record of signs of durable loss of neurons in approximately 10 per cent.; but I do not regard this as a true index of the incidence because patients revealing such neurological signs are more likely to be passed to euthanasia than to the consultant.

Additional to the 58 cases, I think a place in this paper should be given to five cases of B.H.S. tonsillitis complicated by coccidiosis (plus hookworms in two of the cases) which were sent to me for autopsy and toxicological analysis. Four were greyhounds, one was a foxhound; their approximate ages varied from one to three years; their clinical histories preceding the "acute" fatal illness were not obtained. The signs of the fatal intoxication appear to have developed very rapidly; in three cases they were ushered in by vomition and confused behaviour. The advent of a hæmorrhagic watery diarrhœa was followed by attacks or fits of tremors, which were followed by one or more epileptiform fits, with rapid weakening, prostration, shallow breathing, subnormal temperature, fæcal and urinary incontinence, coma and death within about twelve to twenty-four hours.

The onset and passing of rigor mortis, also the onset of putrefaction, were unusually rapid. An intense congestion of all tissues was superimposed on the autopsy findings already recorded for spasmophilia. In the heart and large vessels the blood was unclotted or very imperfectly clotted; traces of liquor sanguinis were found in the serous cavities and tissue spaces. The engorgement of the organs was most evident in the swollen liver, from the cut surface of which a dark blood oozed. The buccal cavity and teeth were covered by a brown slime due to capillary hæmorrhages. Numerous hæmorrhages were found in the mucosa of the fundus and pylorus of the stomach and of the duodenum. The contents of the small intestine mainly comprised watery mucus, altered blood and tissue detritus, having a chocolate colour; those of the rectum varied from a very dark brown to a green colour and from a paste-like to a fluid consistency. Histologically all tissues showed a pronounced hæmolysis; in blood smears the white cells retained their structure and staining properties, but very few red blood corpuscles were recognisable. Capillaries were choked with the debris of red blood corpuscles and blood pigment. The liver showed acute necrosis; the kidneys showed pyæmic nephritis.

To me the interest of these five cases is not confined to their being conjoint infection of the B.H.S. and a bowel parasite terminating in uræmia. From the autopsy findings I feel safe in infering that the hounds had been infected for many days or weeks before the fatal uræmia; but in the absence of the precedent clinical evidence, I can offer only a provisional suggestion that these cases are a "malignant" type of the spasmophilia which we are discussing. Should the collection of detailed histories and clinical evidence of such cases lend support to my suggestion, they might help the biochemists in determining how and by what agency uræmia is produced and its relationship to, if not identity with, that which precipitates a spasmophilic fit.

Hypothesis

The illness (spasmophilia), which is due to conjoint infection by the beta hæmolytic streptococcus (B.H.S.) and a bowel parasite, is an intoxication or poisoning which predisposes to, but does not precipitate, fits of involuntary contractions of muscles.

The precipitation of fits is unpredictable by clinical observation, but it has been observed in some spasmophilic dogs which were injected subcutaneously with vaccine, meat broth or milk.

I imagine a *fit precipitating substance* is produced by the poisoned tissues.

If the fit precipitating substance is produced in excess of its neutralising substance a fit is precipitated; if neutralisation is effected within about two to three minutes the fit ceases; whereas if neutralisation is not effected, death ensues.

Acknowledgment

I gratefully acknowledge the help over many years of my colleague, A. B. Orr, B.Sc., M.R.C.V.S., and Denys Danby, M.R.C.V.S., who also made possible the invaluable follow-up studies; also the generous collaboration of many friends in private practice.

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HUMAN TUBERCULOSIS OF BOVINE ORIGIN
Its Prevention is Still an Urgent Problem

By SYDNEY GORDON TIPPETT, M.B.

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As a doctor interested in the prevention of tuberculosis and, in particular, of that form of this disease due to infection by the bovine type of this tubercle bacillus, I want to see the eradication of bovine tuberculosis in our cattle completed as quickly as possible and within a fixed period of time.

After many years of effort, only 16 per cent. of our cattle herds are to-day certified as free from tuberculosis, so the Minister of Agriculture has recently stated.

The problem of human tuberculosis of bovine origin and its solution must be looked at both from the human side and also from the farmer's point of view.

What are the facts known to the special societies of doctors who interest themselves in human tuberculosis, its treatment and, naturally, its prevention, on this problem? Some 15 years ago the People's League of Health set up a special enquiry to learn what was the danger to human beings from milk containing living germs of tuberculosis. In their report is the following:

"About 2,000 deaths in England and Wales, mostly in children, occur annually from this cause.

"At least 4,000 fresh cases of bovine infection develop each year, an immense amount of suffering, invalidity and often permanent deformity being thus caused by this bacillus."

Following this report, it would be interesting to learn what was done about it by the medical profession, so as to put a stop to this annual great loss of life and which could have and should have been prevented.

Unfortunately, whatever steps were taken in this country they have been proved to be of little practical value in the prevention of human tuberculosis of bovine origin.

Recently the Prophit Survey of Tuberculosis has reported the fact that some 1,500-2,000 persons die each year from tuberculosis of bovine origin. The report does not mention the probable number who get infected and suffer but do not die.

In view of what has been done in the U.S.A. to prevent this form of tuberculosis, the whole of the medical profession must take blame for their apathy and failure to make the public realise this danger to health and to inform them that it could and must be prevented. It is a tragedy that in this country we have failed to follow the work which had been carried out in the United States and so prevent those many thousands of lives, mostly children, being lost.

This is clearly shown by this further statement in the Prophit Survey: "In the U.S.A. results of eradication of bovine tuberculosis and pasteurisation of milk provide definite proof that, in this respect, much tuberculosis in humans is directly and immediately prevented."

This report only referred to the 1,500-2,000 deaths from this form of tuberculosis, but it must also include the thousands who are infected but do not die.

Francis, in his very careful survey of bovine tuberculosis, records his confirmation of the great success of what has been done in the U.S.A. The public has every right to demand a clean and safe milk for everyone.

Unfortunately, milk has been found at times to convey certain other diseases besides tuberculosis, so bovine tuberculosis should be eradicated and all milk for human consumption pasteurised. This is what has proved such a striking success in the U.S.A. and should be done in this country.

There can be no illness or death from tuberculosis at any age except among persons infected by the tubercle bacillus.

We must prevent human beings from infection by the bovine type of tubercle bacillus, and it can be done. It is also quite true to say that probably half the cases of tuberculosis of non-pulmonary conditions are due to infection from another human being and a short reference to work on this is noted by Dr. Jordan at the end of this article. In 1933[1] it was stated: "The medical profession has not so far advocated any measure that has had the slightest effect in reducing the number of persons infected."

How little has been done since then is shown by this Prophit Survey, which found that in the U.S.A. only 10 *per cent.* of young adults now react positively to the tuberculin test, instead of the corresponding 85 *per cent.* in this country, and what this means is clearly brought out by the fact that, "In England and Wales it [tuberculosis] accounts for more deaths between the ages of 15 and 24 than any other single disease and about half the deaths due to all causes."

It follows that if we can prevent human beings from infection by bovine tuberculosis we shall make a definite reduction in the number of deaths from tuberculosis and also prevent thousands each year from being infected who do not *die*.

Now look at this problem from the farmer's point of view.

John Francis[2] states: "According to the most reliable estimate, 20 per cent. of cattle in England are tuberculous, 7.5 per cent. in Wales, 14 per cent. in Scotland, and 17-18 per cent. in Great Britain. The incidence in dairy cows is probably 30-35 per cent. The disease has been practically eradicated from the U.S.A."

Francis, in his very careful survey, draws attention to this probable cause of the high incidence among our dairy herds in this country: "All the evidence suggests that it is the life in the cowshed, when animals are exposed to the intensive droplet and dust infection, that is responsible for the high incidence in dairy cows." With this picture of the danger of the indoor life in the cowshed, it is interesting to note that in the case of sheep which live out of doors all the year round, tuberculosis is practically unknown. Francis quotes further:

"Melvin estimates that the milk production of tuberculous cows was reduced by 10 per cent., and if this figure is accepted it would imply a loss of £3,000,000 per annum in milk yield alone.

"It is a common experience of veterinary surgeons that the usual clinical ailments of cows are less frequent in attested than in tuberculous cows and there can be little doubt that tuberculosis is responsible for much disease which the farmer does not attribute to this cause, and the total loss can hardly be less than £2,000,000 per annum. Quite apart from the economic loss to agriculture and the loss of food to the nation, infection by tubercle bacilli of the bovine type causes ill-health and death in man.

"The thousand or two thousand deaths which occur annually due to bovine infection differ from all other deaths due to tuberculosis, in that they are readily preventable.

" Even when bovine tuberculosis has been eradicated, pasteurisation may still be essential for the control of other milk-borne infections, and it should be realised that the major reason for the pasteurisation of all milk sold by the large dairy firms in the cities is that the time taken in distribution is so long that without this precaution nearly all the milk would be sour before it reached the consumer."

In the eradication of bovine tuberculosis it is obvious that in the future no reactor should be allowed to be taken to the open market and sold there, but all reactors should be killed as it is impossible to tell the extent of the tuberculosis from the tuberculin test when it is positive. The animal may look healthy and have no clinical signs, yet post-mortem examination may disclose extensive disease to be present.

Francis states: " Britain was one of the first countries to adopt the classical method of ' stamping out ' animal diseases by the slaughter of affected animals, and rinderpest, pleuropneumonia, sheep-pox, rabies and glanders have been eradicated. This same method has not been adopted against tuberculosis.

" The attested herds scheme was introduced in 1935, much valuable experience has been obtained and nearly 800,000 cattle (10 per cent. of the total) are now in attested herds.

" When eradication work is being carried out, the danger of infection from man must not be overlooked, and there are several records of those with bovine-type infection causing devastating outbreaks in tubercle-free herds."

Now, in the U.S.A. all this work of eradication of bovine tuberculosis has been in progress for a great many years and it is possible to learn some useful facts about its value.

Dr. J. A. Myers[3]: " Thus, the rapid decline in the incidence of tuberculin reactors among children of this country in the past 10 or 15 years is probably due more to the protection you have afforded them against the bovine type of tubercle bacillus than any other single factor.

" The control of tuberculosis in cattle, therefore, has markedly reduced the incidence of tuberculous infection, the morbidity and the mortality from tuberculosis in man."

It will be at once realised that eradication bovine tuberculosis will cost a lot of money, so it is interesting to learn what was the average cost of testing cattle in the U.S.A.

Dr. J. A. Myers[4]: " Average cost of testing by Bureau Inspection, including salaries and expenses of field veterinarians, was:

46 cents in 1922; 57 cents in 1921; 30 cents in 1924."

There is an interesting piece of advice on education and legislation.

" It is not the veterinarians that are going to accomplish the work entirely, it is not livestock sanitary boards; but the livestock owners themselves.

" It is believed that the best way to have this work introduced into a circumscribed area is to hold meetings and put the matter clearly before the people as to every step it will be necessary to take in order that success may be attained. If the people are not desirous of taking up the work under these conditions, it is best to leave them alone, until such time as they can see their way clear to co-operate.

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"Probably the most effective method of educating the cattle owner was to perform the post-mortem examination of their cattle after they had been shown the tuberculin reaction. Often farmers selected the apparently sleek and well-conditioned animals, but upon viewing the badly diseased organs they were won over.

"It cannot and will not be eradicated or ever controlled by any compromise measures.

"The medical profession was still drawing a sharp dividing line between Tb. infection and Tb. disease, when, in reality, there is none.

"By 1930 it was estimated that the control of tuberculosis among the cattle had increased the returns to dairy farmers to $500,000,000 annually.

"The opposition offered to the control of tuberculosis in the U.S.A. is one of the best demonstrations in the history of the world that science and truth ultimately prevail.

"Human medicine is 40 years behind veterinary medicine in the tuberculosis control programme. The views and activities of the former in 1940 are the same as the veterinarians' in 1900. There has been a strong tendency among physicians in human medicine to fail to recognise the existence of conditions which they cannot see directly or indirectly, although ample evidence is available to prove that such conditions exist."

In the U.S.A. there are now millions of records of the results of post-mortems undertaken on reactors, and they confirm that the diagnosis of tuberculosis is correct in approximately 97 per cent. of the reactors.

The success of the eradication of bovine tuberculosis in the U.S.A. was so striking that when Dr. Lewis S. Jordan in 1947 published his work on school children from 1930-1947,[5] he instituted a similar but modified programme.

In 1930, 13.9 per cent. of school children reacted to tuberculin. In 1946 and 1947, it was only 2.7 per cent.

From the above it will be seen that we can learn from the Americans the great advantage to the country by eradicating bovine tuberculosis.

The farmer will gain by having healthy stock, which will pay him better, and the public will also gain by saving those thousands who die yearly from bovine tuberculosis.

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ACUTE ARSENICAL POISONING IN CATTLE

By K. G. TOWERS, B.V.Sc., M.R.C.V.S.,
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DURING the early winter of 1947-48 our attention was drawn to a number of herds in a district in Yorkshire: in each the cattle suffered from a sudden onset of diarrhœa, together with a severe drop in milk yield in the lactating animals. In Yorkshire we have seen quite often outbreaks of diarrhœa of

unknown ætiology resembling that described by Rollinson (1948), especially in dairy herds which have been recently housed for the winter months. In this series of cases, however, the condition did not spread rapidly through the herd, but the animals were simultaneously and suddenly affected. The illness was serious when the loss of milk and, in one herd, the deaths of half of the animals are taken into account.

The first herd was seen in September, 1947, when the milking stock of 20 animals was suddenly affected. All showed sudden severe watery diarrhœa which did not contain blood, and one animal died after two days' illness. A post-mortem examination was carried out, but no significant changes were seen and cultural examination of fæces, mesenteric glands and other internal organs failed to detect a bacterial infection. Agglutination tests were carried out on the blood of a few cows in the herd, but no significant titres against Salmonella antigens were recorded. The suddenness of the attacks hardly suggested Johne's disease and the fæces of the live animals were not found to contain *M. paratuberculosis*.

The only suggestive line of enquiry seemed to be the feeding of the animals. The water supply and the grazing appeared to be above suspicion, but in the byre a new consignment of an unrationed "locust bean meal" had been included for the first time in the rations supplied at the milking immediately prior to the illness. This type of meal had been fed on sundry occasions in the past without causing trouble, and subsequent inquiry showed that the local agent had distributed a considerable quantity of the food without any illness being reported. The meal was subjected to botanical analysis and was found to be a mixture of mainly yellow and white particles, which appeared to be pure locust bean meal. This eliminated the possibility of castor seed contamination. Samples of this meal and the other foods used on the farm were fed to laboratory animals without causing illness. The milk yield, which in a day had practically ceased, slowly improved, and after a month the animals were normal except for one cow, which was still inclined to stagger when walking. No diagnosis was made and the cause of the trouble only became apparent after the next cases. The components of the ration fed to the cows were then collected and sent to Dr. Green, Weybridge, who reported that no abnormal amount of arsenic was present.

The next case occurred in October on a farm seven miles away from the first. A herd of 16 animals, all in milk, was normal in every respect at the morning milking on a particular day. At the evening milking they were all more or less severely ill, passing copious amounts of green watery diarrhœa and the quantity of milk obtained was no more than one or two pints, given by each of the heaviest milkers. At no time was blood seen in the fæces of these animals and there was no excessive straining. Some slight abdominal pain was shown and in 48 hours two animals died after becoming so exhausted that they could not rise. A further two animals developed symptoms of excitement, which increased to mania, and they were shot on the third day. By the fourth day a further three milk cows died quietly from exhaustion, the last death occurring a week after symptoms started. At no time during the

illness did any of the herd show a rise in temperature; in fact, some were subnormal.

The herd had been grazing aftermath for the last 10-11 weeks and the field was quite clean, being surrounded by dry stone walls on three sides and a wire fence with hedge on the remaining side. There was thus no access to poisonous plants, neither was there any sign of rubbish having been thrown into the field. The water supply was from a spring and appeared wholesome. Post-mortem examinations were carried out on the first four animals, and the two which were destroyed presented a perfectly normal appearance. In the two which died there was an extensive, but by no means intense, reddening of the carcase and internal organs. The subcutaneous tissue seen in the flayed cadaver was injected, as also was the omentum. There was a mild diffuse enteritis, which was most marked in the small intestine, and a reddening of all the stomach walls, which was most obvious on the internal aspect of the rumen. Some œdema was present in the folds of the mucous membrane of the abomasum. The liver and kidneys appeared slightly pale and yellowish, and were examined by a "spot" test for arsenic, but were reported negative. The contents of the small intestine were of a thin, bloody nature; they were filtered and injected intravenously into mice, without causing death in two days. The suddenness of the appearance of symptoms and the lack of fever did not suggest an infectious agent, so it was not surprising when cultural examination proved negative. The alimentary inflammation, which was not seen in the mouth or œsophagus but which was present in the rumen, was thought to be caused most likely by an irritant poison.

As in the previous case, it was discovered that a new batch of foods had been received from the local supplier, who was not the agent concerned in the first case, and only one feed of this new batch had been fed in the morning of the day when symptoms started. Another point throwing suspicion on the food was that three stirks received a bucket of provender and the greediest feeder, which might be expected to have had the major share, developed the most acute symptoms. These new rations included dairy mixture, oats mixture and locust germ meal. These components of the mixture fed to the herd were fed separately to laboratory animals and failed to cause illness.

The mixed prepared ration as fed to the cows was sent to Dr. Green with a request that it should be examined for irritant poison; he reported the presence of arsenic in considerable quantity. The individual components were then sent for analysis and the oats mixture alone was found to contain arsenic in the order of 0.1 per cent. As_2O_3. This mixture had been made on the farm from oat husks, pea meal and locust germ meal. None of these components was found to contain arsenic, on subsequent analysis.

Material from the post mortems was now sent to Weybridge and was reported on as follows:—

The cow which died:

Liver, 26 p.p.m. arsenic expressed as As_2O_3
Kidney, 17 „ „ „ „ „

The cow which was shot:

Kidney, 13 p.p.m. arsenic expressed as As_2O_3

Dr. Green commented that these tissue values left no doubt about diagnosis.

During the investigation into this case, a third farm was found where the now well-known syndrome of illness and diarrhœa in all the herd was encountered. This farm had also received fresh rations delivered by the same supplier and on the same day as the second case above. This farm had been well supplied with food, so that these new rations were not used for several days and then were well mixed and diluted with other food before being fed. The result was that no deaths occurred and symptoms were not so severe. The mixed ration as fed to the cows was found to be strongly positive for arsenic, but again the components of the ration proved negative on analysis.

It is noteworthy that one of the constituents was the unrationed locust germ meal.

Inquiries were made at the supplier's end, but no other complaints had been received and the suspected food had been issued for some time and fed on numerous farms without the occurrence of any illness.

In January, a fourth farm was found where two animals died and the others showed the sudden diarrhœa which was now associated with poisoning. The carcases at post-mortem examination were slightly reddened but no other anatomical abnormality was detected. The livers were forwarded to Weybridge and the arsenic content, expressed as As_2O_3, was reported as 10 and 12 p.p.m. This farmer had received a supply of "S" meal or "cocoa bean meal" which was similar in appearance to the locust bean meal of the previous cases, and he had fed only two feeds containing this meal when his milk yield fell almost to nil. The remainder of the ration had been fed for some time without ill-effect. All the cows had received a similar amount of the suspected meal, but the symptoms varied from death to slight illness, and especial care was taken to obtain a representative sample of the meal for analysis. Dr. Green reported that this meal gave a strongly positive qualitative test for arsenic.

Attempts to trace the origin of this meal, which was common to all four outbreaks, have failed to show a common source. No other cases have been reported to the mills supplying the local agents. A possible method of contamination of the meal, either during or after delivery, has not been found. Sheep are kept on all the farms but are taken elsewhere for dipping and sheep dip is never stored. The source of the arsenic, therefore, has not been discovered.

Discussion

It is probable that the contamination of the meal was present in a localised and patchy distribution, which would explain the early negative analytical results. The faulty "spot" test again delayed diagnosis. Fatty degeneration of the parenchymatous organs is not to be expected in acute arsenical poisoning, but it is noteworthy that the only yellow livers and kidneys discovered were present in animals dying quickly after poisoning, and in those which lived some time—

up to one week—there were no fatty changes apparent at post-mortem examination. Attention is also drawn to the dramatic cessation of milk production in the affected animals. (Two out of the four farmers remarked that for some time after recovery the milk was thick and creamy.) Because of sampling errors which are unavoidable when sampling foodstuffs, no accurate estimation can be made of the quantity of arsenic received by individual animals, but of the food containing 0.1 per cent. As. approximately 1 lb. was fed; this gives a figure of approximately 0.5 grms. arsenic reckoned as As_2O_3, but this presupposes a uniform contamination of the food. The abdominal pain said to be associated with acute arsenical poisoning was not a feature of these cases. The irritation of the poison did not cause salivation and the staggering and weakness must be directly ascribed to the severe purgation.

No treatment was given in any of these cases. The prompt withdrawal of the suspected food was followed by recovery, except in the severely affected cases which died before assistance had been obtained in the last outbreak.

Acknowledgments

Thanks are due to Mr. Leslie, M.R.C.V.S., with whom these cases were seen; to Dr. Green and the Biochemistry Department at Weybridge for the analytical work; and to Mr. P. L. Shanks, B.Sc., M.R.C.V.S., for his interest and assistance.

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LEUCOTROPIN

In the Treatment of Painful Arthritic and Other Lesions

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Hendon

LEUCOTROPIN is a combination of hexamine and phenylcinchoninic acid dissolved in a 15 per cent. solution in distilled water.

The solution was tested in a laboratory for any bactericidal action.

Whilst it was negative on streptococci, it was found that it produced a slight clear ring in cultures of Coli bacilli.

Leucotropin was tried in the following cases:—

(1) *Spaniel, Dog, eight years old*

The animal was brought to the surgery at the beginning of August with a streptococcal infection under the right eye and a massive bilateral tonsillitis. He was treated with penicillin and sulpha drugs, but the infection was sluggish in the reaction to treatment.

Ten days later the dog showed every sign of a polyarthritis subsequent to the streptococcal infection. The animal was unable to walk, it could not open its mouth more than about a quarter of an inch, and it was in considerable pain.

There was a distinct crepitus in the temporo mandibular joint, so much so that a fracture was suspected. The part was X-rayed. No abnormality was seen.

The animal was treated for a few days with aspirin and pethidine, but did not improve and began to vomit.

Leucotropin treatment was then started, the dosage being increased from 1 c.c. on the first day to 3.5 c.c. on the third day in daily intramuscular injections. The injections were painful and a local anæsthetic was added to overcome this drawback. At the level of 3.5 c.c., the dog stopped vomiting and began to move slowly, though painfully. It was still impossible to open the mouth.

The injections were then given subcutaneously and as this method proved painless the local anæsthetic was discarded and the amount given increased to 7 c.c. This stage was reached on the fifth day. For the following eight days, 7-8 c.c. were given subcutaneously.

On the seventh injection, the spaniel began to walk again and to eat of its own accord. After the thirteenth injection an intermission of a week was made and treatment restarted again after this week's period of rest.

The animal received another twelve injections. After the third day of the second period, the patient began to play and has made an uneventful recovery almost to his normal state. His mouth was then completely free and could be opened to the full extent. Appetite was good.

Frequent urine tests made during the period of treatment demonstrated a large amount of salt-excretion, but neither albumen nor bile-salts. The heart and pulse remained normal throughout the treatment.

As the animal's health seems to be subject to changes in the weather, the owner has been provided with leucotropin tablets, one to be given morning and evening whenever signs of arthritic pains are shown.

(2) *Bullmastiff, Dog, two years old*

One and a half years ago this animal was treated for severe rickets. His four legs, especially the hind ones, were deformed.

Four weeks ago I was called in and found that the animal was suffering from a rheumatoid arthritis of the hind legs. The dog received 10 injections of 10 c.c. leucotropin solution subcutaneously; they were given at two-day intervals. The animal began to walk on the third day and returned to its normal state after the tenth injection. He seems to benefit also on leucotropin tablets, which he now receives twice daily. Urine test showed salts but no other abnormalities. The appetite improved immediately after the first injection.

(3) *Kerry Blue, Bitch, eleven years old*

The diagnosis was an arthritis of the left shoulder, caused by an accident. The animal refused to put the foot down to the ground and was in such pain that a fracture was suspected. An X-ray was taken and was negative. Salycilates or pethidine did not improve the condition.

After the sceond injection of 6 c.c. of leucotropin the animal was completely cured.

(4) *Airedale, Dog, eleven years old*

This animal was brought to the surgery because of obvious pain in the lumbar region. Urine tests were positive for albumen.

One injection of 6 c.c. of leucotropin was given. Within an hour the animal relinquished its tense and hunched-up position. Leucotropin tablets twice daily have kept the outer appearance of the dog normal, but the amount of albumen in the urine was not reduced.

(5) *Peke, Dog, two years old*

The animal was brought to the surgery with the well-known syndrome of lumbago, i.e. stiffness, abnormal gait and shrieking when touched. One injection of 5 c.c. was sufficient to cancel the pain and abnormal behaviour.

(6) *Manchester Terrier, Bitch, eleven years old*

Because of its combination with hexamine, leucotropin was tried in this case of chronic nephritis. Fifteen injections of leucotropin were given. The amount of albumen in the urine seemed only slightly reduced, although the thirst was decreased and the general behaviour of the animal seemed happier.

(7) *Spaniel, Dog, six months old*

The animal had been treated at a clinic for cystic calculi. He was suffering from a very painful cystitis and acute albuminuria, and had had several fits.

After the first injection of 5 c.c. leucotropin, the pain seemed to disappear and no more fits occurred. After the fourth injection the animal's urine and behaviour were normal.

Summary

The above seven cases were treated exclusively, at one stage or another, with leucotropin.

Apart from these, several other cases were treated but they have to be classed as inconclusive as penicillin and other drugs were used in addition to leucotropin.

It seems that leucotropin is of considerable value as an analgesic in arthritic, rheumatic, and nephritic pains. It has not been detrimental, as far as could be ascertained, to liver or kidneys. It alleviates suffering without any hypnotic by-effects.

The action begins one to two hours after administration and lasts at the first injections for 4-6 hours. After several daily applications the action seems to be prolonged to eight hours.

It is doubtful if leucotropin has any disinfectant or healing action in chronic nephritis or infectious diseases. Its beneficial action lies in its analgesic effect, as adjuvant to established treatment.

The injections applied subcutaneously are painless and the subsequent treatment with tablets well tolerated.

I am obliged to Messrs. Silten, Ltd., of Hatfield, for providing the leucotropin in these trials.

Addendum to Mr. Lustig-Lendva's Article by J. McCunn

Through the courtesy of Mr. Lustig-Lendva I have been enabled to put leucotropin to test on osteoarthritic lesions in the horse. Two horses were subjected to treatment, a grey hunter and a black show hack.

In both cases the osteoarthritic lesions were in the corono pedal joint following puncture of the foot by a foreign body.

The hunter picked up a nail. It penetrated to the region of the joint. When admitted to hospital (4/11/48) there was a month's history of injury followed by extreme lameness and pain. Suitable treatment had been applied by the owner's veterinary surgeon. Infection and suppurative reaction had been controlled. There was acute pain and lameness, the animal being scarcely able to put the point of the toe on the ground. After three weeks of treatment in hospital no change was registered and I had grave doubts as to ultimate recovery.

At this stage leucotropin was injected subcutaneously each day and for five days. The amount injected was 10 c.c., 20 c.c., 30 c.c., 30 c.c., 30 c.c. There was some degree of local reaction manifested by pain and swelling.

On the morning after the first injection I detected an improvement. This improvement was quite marked and it was progressive as the injections proceeded. By the fifth day the clinical picture was completely changed and although some degree of lameness persisted the animal was able to walk, placing the foot firmly on the ground, and was fit for discharge from hospital. It was discharged on December 13, 1948. The lameness continued to improve and has completely resolved with no further treatment. It is interesting to note that X-ray photographs of this horse's foot, taken just previous to the administration of leucotropin, demonstrated a considerable degree of erosion of the navicular bone. This erosion extended from the lower border of the bone to a point just above the mid-transverse line.

The hack trod on a broken bottle on August 8 whilst at a show. It had been under competent veterinary treatment until admitted to hospital on October 27, 1948. On admittance there was intense lameness and pain in the rear hind limb. The point of puncture was seen easily and from it there exuded an oily pus. X-ray pictures were taken and, as far as one could judge, the bones were undamaged and no foreign bodies were seen. As the radiologist informed me that certain varieties of glass did not show on X-ray, it was decided to explore the lesion surgically. Under general anæsthesia, the frog and other tissues were stripped and the plantar apeneurosis exposed. No foreign bodies were found. The wound was packed with penicillin and procaine, and this was also injected via the heel to the navicular region. The procaine showed an effect which lasted for a few hours, but then the pain and lameness returned. There was no further response to our treatment and I was considering advising destruction. On December 1 the first injection of leucotropin was given. The dosage was as on the grey hunter (treatment exactly coincided in dates and dosage).

The result of treatment was equally as favourable as in the case of the hunter, in fact this animal was actually discharged to home on December 7.

If one horse only had been involved, I think that one might, with good reason, say that the recovery was probably the end result of previous long-continued treatment; in other words, recovery only happened to be coincidental with the administration of leucotropin. But here are two horses, suffering from exactly the same type of lesion and which had been under competent veterinary treatment for a considerable time, without any alleviation of symptoms. Treatment had failed to elicit any beneficial or even hopeful response. In both cases improvement was immediate and progressive, and this very satisfactory result coincided with the administration of leucotropin.

I shall certainly put it to further trial and test as occasion arises.

XIVth INTERNATIONAL VETERINARY CONGRESS

Once again it is the great privilege of the veterinary profession of this country to provide the venue for an International Congress and to entertain colleagues who will attend that Congress from overseas.

The staging of such a Congress is no mean task and the success and value of such an affair depends greatly upon the energy and goodwill of those whose duty it is to act as host.

A Congress such as this may serve two purposes: first, the dissemination of knowledge in order that our science may be advanced, and second, by establishing social and other contacts we may foster that feeling of mutual goodwill and trust which is so necessary in the sphere of international relations.

We have great pleasure in placing before our readers the preliminary announcement of the Executive Committee, and it is our sincere wish that the profession in this country will respond in such a manner that this Congress will be an even greater success than was the one held in London in 1930.

Object

The object of the International Veterinary Congresses is the advancement of the science and practice of veterinary medicine and surgery. To this end discussions are arranged on what are considered to be the most important scientific and practical questions of the day, with a view to their solution by a mutual interchange of opinions and personal experience.

Programme

As it is considered that the present world food situation is the most important scientific and practical question of the day, the Congress programme has been based on this theme. Every morning there will be a plenary session at which a paper will be presented by some person of world reputation on

the veterinary profession's contribution to some aspect of world food production. Sectional meetings will be held in the afternoons. The programme for these is designed to cover a wide range of subjects, and, whatever the speciality of any member of the Congress, he should find papers of direct interest to him.

Executive Committee

The President . . .	Sir Daniel Cabot
Vice-President . . .	Professor J. B. Buxton
Honorary Secretary . . .	Dr. W. R. Wooldridge
Honorary Treasurer . . .	Mr. G. N. Gould
General Secretary . . .	Mr. W. G. R. Oates
Organising Secretary . .	Lt.-Col. J. A. Gordon Roberts

To whom all communications should be sent at:

10, Red Lion Square, London, W.C.1.

Administration

1. Place of Meeting

The meetings will take place at the Central Hall, Westminster, and in Church House, Westminster, London, S.W.1.

2. Opening Date

The Congress is under the patronage of His Majesty the King, and it is hoped that His Royal Highness the Duke of Gloucester, who is the President of the Royal Veterinary College and Hospital of London, will be able to open the Congress.

The first session will take place at 10 a.m. on Monday, August 8, 1949, and the Congress will last until Saturday, August 13, inclusive. Registration of members will take place at Central Hall on Sunday, August 7, from 2 p.m. (1400 hours) onwards. Certain social functions will take place on the evening of Sunday, August 7.

A detailed programme showing the times at which the General and Sectional meetings will take place, giving the names of speakers and titles of papers, will be published in the official programme to be issued later.

3. Social Functions

Many official and other social functions will be held during the week of the Congress, at which delegates will be able to meeet their confrères from other countries. Details of these functions will also be notified in the official programme.

4. Excursions

During the week August 15 to 20, visits will be arranged to places of interest to veterinarians, such as: Veterinary research stations, veterinary col-

leges, abattoirs, breeding and racing establishments, etc., etc. Every effort will be made to show and to give information about the latest developments in veterinary research.

Tours will also be arranged to places of interest and beauty spots in the British Isles.

5. EXHIBITION

An exhibition of the latest instruments, appliances, laboratory equipment, drugs, disinfectants, antiseptics, books, etc., etc., will be arranged in premises convenient to the Central Hall. Admission to members will be free.

6. MEMBERSHIP

Official invitations have been issued by His Majesty's Government to Foreign, Commonwealth and Colonial Governments requesting the sending of delegates to the Congress. Representation has been invited not only from Government veterinary departments, but also from universities, veterinary schools and other interested bodies in every country.

Application for membership should be made direct to the Organising Secretary, 10, Red Lion Square, London, W.C.1. All applications should be accompanied by a remittance for the exact amount of the subscription. Cheques, Money Orders, etc., should be made payable to the "XIV International Veterinary Congress." Names, titles and addresses should be clearly written, preferably in block letters.

Early application for membership is requested.

7. SUBSCRIPTIONS

Ordinary Members—£4 (four Pounds) Sterling, which includes Congress badge, entertainments (except Congress dinner, for which a charge will be made) and one copy of the full report of the Congress.

Student Members—£1 10s. (one Pound, ten Shillings) Sterling, which includes Congress badge and the right to attend meetings, but does not confer the right either to speak or vote or to receive a copy of the Congress report. The report may, however, be purchased separately, at a price to be notified later.

Associate Members (wives and families of members)—£1 (one Pound) Sterling, which includes Congress badge, the right to attend special functions which will be arranged for them and the use of special accommodation for social purposes.

8. TRAVEL ACCOMMODATION

Messrs. Thomas Cook & Son Limited, of Berkeley Street, London, W.1, have been appointed travel and accommodation agents to the Congress.

Hotel accommodation in London during August is likely to be heavily booked, and prospective members are advised to make early arrangements.

If assistance in booking hotel accommodation is required, members should get in touch with Messrs. Thomas Cook & Son direct.

It is hoped that it will be possible to obtain some travel concessions on the British Railways for members travelling to London for the Congress, details of which will be notified later.

9. ENQUIRIES

All enquiries and communications should be addressed to:

Organising Secretary,
XIVth International Veterinary Congress,
10, Red Lion Square, London, W.C.1.

Telegraphic Address: CENTAURUM, HOLB, LONDON.

Telephone Number: HOLborn 3584.

REVIEWS

" FARMER AND STOCK-BREEDER YEAR BOOK, 1949," 73rd Edition. Published on December 15, 1948, by *Farmer and Stock-Breeder,* Dorset House, Stamford Street, London, S.E.1. Price: 7s. 6d. (postage 6d.). Size: D8vo (8¾ in. x 5½ in.). 340 pages, over 130 illustrations. Quarter bound, paper boards.

Following up its successful post-war reappearance last year, " Farmer and Stock-Breeder Year Book " continues to provide the friendly help which has always been associated with it. As in previous years, it makes a wide appeal to many farming interests.

" Farmer and Stock-Breeder " photographs, which illustrate the special photogravure section of prize-winning animals, will be as popular a feature as ever. Associated with this there is again a summary of prices of pedigree and commercial stock which will continue to be of value for reference purposes. The Buyer's Guide and the numerous lists of names, addresses, telephone numbers and other pieces of information provide, at a glance, just the kind of detail that no one wants to carry in their head, but which is always useful to have on their desk.

FAT LAMB PRODUCTION. Boaz, T. G., Bywater, T. L., Robertson, C. C. A., University of Leeds and Yorkshire Council for Agricultural Education. Bulletin No. 211.

SWALEDALE ewes were crossed with Border Leicester, Suffolk and Wensleydale rams with a view to ascertaining the relative efficiency as mothers, for fat lamb production, of the resulting first-cross ewes when mated with Hampshire rams. The results obtained in eleven successive seasons are recorded and analysed. In terms of gross return per ewe bought on the farm and mated initially, the Suffolk-Swaledale cross gave the highest return of £17 18s. 5d.

XUM

per ewe, the Wensleydale-Swaledale cross gave a return of £15 4s. 7d. per ewe, and the Border Leicester-Swaledale gave the lowest return of £14 3s. 8d. per ewe.

It is shown that the difference in results are primarily due to variations in longevity of the ewes, fertility and rearing ability. Whilst the Border Leicester-Swaledale crosses lambed down at an average rate of two lambs per ewe, there was a fairly high incidence of barren ewes and losses amongst ewes. The Suffolk-Swaledale crosses produced slightly fewer lambs per ewe, but losses and barrenness amongst ewes were less so that more lambs were born alive and more reared. The results in the groups of Wensleydale-Swaledale crosses show a variability which is ascribed to differences inherent in the two Wensleydale rams whose progeny are compared; nevertheless, efficiency again seems to be related more to the wastage among and longevity of the ewes rather than to their ability to produce and rear lambs.

G.A.W.

MODERN POULTRY HUSBANDRY, by Leonard Robinson, N.D.A., London: Crosby Lockwood and Son, Ltd., 1948. 21s.

THIS book by the Editor of the well-known weekly, "Modern Poultry Keeping," is almost a model of its kind; it does exactly what it sets out to do, namely, "to convey to the reader some conception of the poultry industry and to explain the fundamental principles of poultry breeding and production." Mr. Robinson compresses into this moderately sized work a wealth of information on the "science" as well as on the "art" of poultry husbandry, and especially do we commend his prefatory assertation that "theoretical knowledge is not a substitute for practical experience, but it is true to say that unless the fundamental principles of the various phases of the work are understood it will not be possible for the poultry farmer to manage his farm efficiently." Did not one of the great scientists of last century, Thomas Henry Huxley, mean the same thing when he defined the practical man as he who practises the errors of previous generations?

All the important aspects of poultry husbandry are adequately covered in this book, which, starting with a concise survey of the past history of the poultry industry in Britain, passes on to deal in separate Chapters with the systems of poultry keeping, breeds and breeding, incubation and rearing, the management of laying stock, poultry nutrition, table poultry production, houses and appliances, and finally poultry diseases, about which subject Mr. Robinson has had the wisdom to first consult an acknowledged veterinary expert in this field before sending his manuscript to the printers.

The whole approach of the book is predominantly one of common sense, and in the main the author sets out his points clearly and in a readable way. In one or two of the Chapters, however, the reader gets something of a jolt from the very diverse nature of the subject matter covered by the main headings of the Chapters in question. Thus, Chapter 7, "Systems of Breeding," first treats of cross-breeding, line-breeding, etc., but after a few pages of discussion on this

theme, which is quite correct and appropriate to the chapter heading, there is suddenly projected into the Chapter a consideration of what the present reviewer holds to be more proper to the subject of reproductive physiology than to breeding systems, namely, " methods of mating," " factors affecting fertility," " duration of fertility," etc. Less eccentric, perhaps, is the inclusion of a short section on poultry manure in the Chapter (17) entitled " The Feeding-Stuffs "; perhaps Mr. Robinson, with or without conscious intention, here emphasises that under bad conditions of husbandry poultry droppings do perhaps constitute in a sense a " feeding-stuff," albeit a potentially dangerous one.

One further matter for criticism: at the end of the Chapter dealing with " Poultry Diseases " there is a short section dealing with sanitation, excellent so far as it goes, but we seriously suggest that when the next edition is under preparation this question of poultry hygiene should receive far more prominence than it does in the present edition; one gets the impression that it has been tagged on more or less as an after-thought to the subject of poultry disease. Surely, as Mr. Robinson himself suggests in the first Chapter, the overriding importance of effective control of the disease hazard, if the poultry industry is to attain an efficient and profitable status, clearly indicates that the correct application of hygienic principles must be a matter of major interest to every poultry farmer. From such a point of view arises our desire to see this particular subject raised to the dignity of a chapter in itself, wherein the information from the section referred to, and also that embodied under other subject headings in various parts of the book, are gathered together at one place in the book, amplified or expanded, and presented in a format which leaves the reader in no doubt as to its very important place in practice of poultry-keeping.

We have no qualms in stating that this book should be useful to every veterinarian who has, or is liable to have, contacts with any branch of poultry husbandry. Equally, it is a textbook which we commend to the attention of every student proceeding to the professional examinations in Animal Husbandry and Veterinary Hygiene.

N. J. S.

NOTICES

£10,000 FOR CANINE RESEARCH

For the second year in succession, members of the National Greyhound Racing Society have contributed £10,000 to the Canine Section of the Animal Health Trust.

In forwarding a cheque to the Trust, Sir Louis Greig, President of the Society, says that in spite of the adverse financial effect of taxation during 1948, the Society's members have maintained their contribution because they regard the establishment of the research station as a national necessity.

They feel that this section is of equal importance in its field as the service

given by the equine, cattle and poultry research stations now in operation by the Trust.

C. J. HEWLETT AND SON, LTD., announce that, in accordance with the Purchase Tax No. 5 Order, 1948, the following are not now subject to tax: Hormonigen Tablets, Hormonigen Elixir, Ampoules of Cardiac Tonic, Hewlett's Sarcogen.

MESSRS. BURROUGHS WELLCOME & Co. announce that the following "Wellcome" brand veterinary products are now freed from Purchase Tax: Canine Distemper Prophylactics Method I and Method II, Canine Anti-Distemper Serum, Pregnant Mares Serum (P.M.S.), C. Pyogenes Anti-Serum, Black Disease Vaccine and Anti-Serum, Contagious Pustular Dermatitis (Orf) Vaccine, Leptospira Vaccine.

REPRINTS

The publishers are desirous of reminding contributors that owing to the slight improvement in the paper supply position, a maximum number of 150 reprints of articles can be supplied, provided they receive instructions when the author returns his corrected proofs for press. These will be produced at a reasonable cost.

Publishers' Notices

THE BRITISH VETERINARY JOURNAL, with which is incorporated THE VETERINARY JOURNAL, is published monthly, and copy for advertisements should be in the hands of the publishers—Baillière, Tindall and Cox—not later than the 20th of the preceding month, if proof is required. Tel.: Temple Bar 3386.

Letters for the Journal, literary contributions, reports, notices, books for review, exchanges, new instruments or materials, and all matter for publication (except advertisements) should be addressed to the Editor.

Annual Subscription, 30s. ($6.50 U.S.A. currency, post free.)